

Analysis of SEIR Model of Ebola Virus through Equilibrium Approach

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ABSTRACT: The Ebola outbreak in Guinea, Liberia and Sierra Leone of West Africa December 2013 has sickened more than 28,610 people and taken more than 11,308 lives, making it the largest Ebola outbreak ever recorded. In order to understand the forces that drive this outbreak, the force of infection must be broken down into the form differential equations. So we present a mathematical model based on the standard SEIR model. The disease-free equilibrium point of the model was established and its stability analysis carried out using the Routh-Hurwitz criteria.

KEYWORDS: Mathematical model, Ebola virus, Disease free equilibrium, Endemic equilibrium.

I. INTRODUCTION

Ebola virus disease (EVD) is a severe, often fatal illness in humans. EVD outbreaks have a case fatality rate of up to 90%. Ebola first appeared in 1976 in two simultaneous outbreaks, in Nzara, Sudan, and in Yambuku, Democratic Republic of Congo [1-2]. There are no licensed specific treatments or vaccine available for use in people or animals. Genus Ebola virus is 1 of 3 members of the Filoviridae family (filovirus), along with genus Marburgvirus and genus Cuevavirus. Genus Ebolavirus comprises 5 distinct species: Bundibugyo ebolavirus (BDBV), Zaire ebolavirus (EBOV), Reston ebolavirus (RESTV), Sudan ebolavirus (SUDV) and Taï Forest ebolavirus (TAFV) [3]. The incubation period of Ebola virus disease (EVD) varies from 2 to 21 days, with an observed average of 8 to 10 days. Human-to-human transmission occurs only via direct contact with blood or bodily fluids from an infected person who is showing signs of infection or by contact with objects recently contaminated by an actively ill infected person [4].

Indirect contact with environment and fomites soiled with contaminated bodily fluids (e.g. needles) may also occur. Air born transmission has not been documented during Ebola outbreaks, however, in February 2015 a group of researchers published a paper suggesting that it is very likely that at least some degree of Ebola virus transmission currently occurs via infectious aerosols but this has never been demonstrated in humans [5].

There is no risk of transmission during the incubation period. The most common symptoms experienced by persons infected with the virus are the sudden onset of fever, intense weakness, muscle pain, headache and sore throat. This is followed by vomiting, diarrhea, rash, impaired kidney and liver function, and at advanced stage, both internal and external bleeding [6]. Laboratory findings include low white blood cells and platelet counts and elevated liver enzymes. The spread of EVD between and within the three neighboring countries accounting for the majority of the cases noted so far – Guinea, Liberia, and Sierra Leone. By nature, Ebola is highly contagious and deadly and there are no drugs or proven Ebola virus-specific treatment at moment. In the light of the foregoing, there is therefore an urgent and serious need to have coordinated responses from all angles in order to combat the disease effectively. As part of this coordinated approach, is the need for mathematical modelling of the disease. Mathematical models have been important tools in analyzing the epidemiological characteristics of these infectious diseases since the pioneer work of Kermack and McKendrick (1927) [7].

In this paper equilibrium analysis has done for disease free equilibrium and endemic equilibrium through Ordinary differential equations using Routh-Hurwitz criteria.

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II. REPORTED CASES AND DEATHS

28,610 confirmed, probable, and suspected cases have been reported in Guinea, Liberia, and Sierra Leone, with 11,308 deaths (Figure 1) since the onset of the Ebola outbreak from 1st March 2014 to 27th March 2016 [8]. From WHO report, due to EVD in Guinea 66.72%, Liberia 45.04% and Sierra Leone 28% peoples have been died out of reported cases. Maximum EVD cases are reported in Sierra Leone and deaths in Liberia.

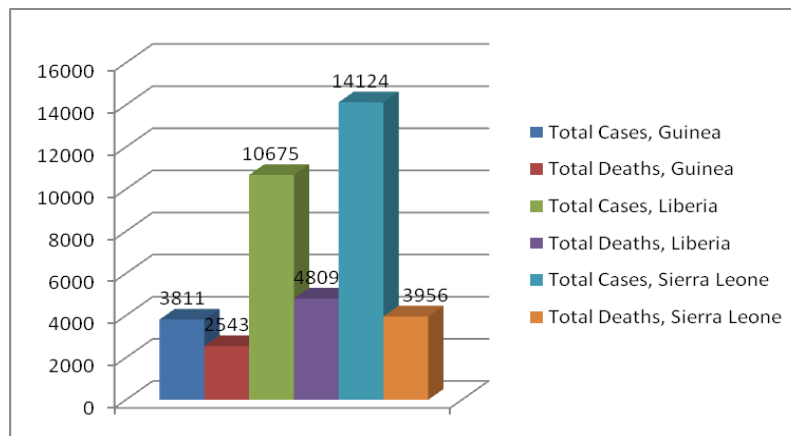


Figure 1. World health organization (WHO) report – 27th March 2016

On 9th June 2016, WHO declares the end of the most recent outbreak of Ebola virus disease in Liberia. Sierra Leone was declared free of Ebola on 17th march 2016, and Guinea on 1st June 2016. But the global health organization cautioned that Liberia, Guinea and Sierra Leone remain at high risk for additional small outbreaks of the disease and must remain vigilant [9].

III. MODEL

The SEIR model for EVD, in which the population is divided into four classes as Susceptible(S), Exposed(E), Infectious(I) and Recovered(R). This model is the extension of SIR model [10] i.e., insertion of exposed classes in SIR model. In this model the Susceptible are the portion of population who are at the high risk of EVD. The Exposed are the individual who infected with EVD with no positive symptoms. The Infectious are the individual who infected with EVD and passes it to Susceptible peoples. The Recovered are the individuals who recovered from EVD.

Assumption:

1. μ =birth rate = death rate
2. Infant should not have immunity against EVD

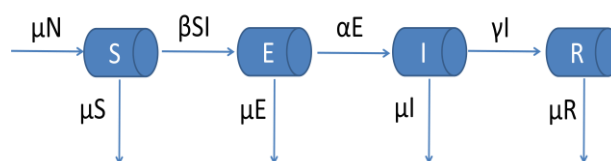


Figure 2. SEIR Model

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(An ISO 3297: 2007 Certified Organization)

Website: www.ijirset.com

Vol. 6, Issue 6, June 2017

The individual welcome to model with μN in S class. The Susceptible individual goes to Exposed class with βSI and Exposed individual once infected then it transfer to Infectious class with αE . Then after the proper immunity against EVD the infected individual goes to Recovered class with γI . The μS , μE , μI and μR are the group of peoples, who pass away from S, E, I, R classes respectively.

Notation:

EVD	Ebola virus disease
S(t)	The population of susceptible individuals at time t
E(t)	The population of latently infected individuals at time t
I(t)	The population of infected individuals at time t
R(t)	The population of individuals who have been treated and have recovered from the infection at time t
α	The rate at which susceptible individuals become latently infected with the EVD
β	The rate at which latently infected individual become actively infected with the EVD
γ	The natural mortality or death rate

Mathematical representation of SEIR model:

The system that governs the differential equation is given by:

$$\frac{dS}{dt} = S' = \mu N - \mu S - \beta IS \tag{1}$$

$$\frac{dE}{dt} = E' = \beta IS - (\mu + \alpha)E$$

$$\frac{dI}{dt} = I' = \alpha E - (\gamma + \mu)I$$

$$\frac{dR}{dt} = R' = \gamma I - \mu R$$

Proposition 1.1. The total population N being modelled by the SEIR model (1) is constant in time, that is, $S(t) + E(t) + I(t) + R(t) = N$ for all $t \geq 0$.

Proof Follows by adding the right-hand side of equations (1): $\frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0$

Now we introduce new variable by assuming $S = Ns, E = Ne, I = Ni, R = Nr$ and the variable R does not appear in the first three equations of (1), so it is enough to analyze the following reduced system:

$$e' = \beta is - (\mu + \alpha)e \tag{2}$$

$$i' = \alpha e - (\gamma + \mu)i$$

$$s' = \mu - (\mu + \beta i)s$$

International Journal of Innovative Research in Science, Engineering and Technology

(An ISO 3297: 2007 Certified Organization)

Website: www.ijirset.com

Vol. 6, Issue 6, June 2017

Now we linearize these equations, we get,

$$\begin{bmatrix} \frac{dE}{dt} \\ \frac{dI}{dt} \\ \frac{dS}{dt} \end{bmatrix} = (F - V) \begin{bmatrix} E \\ I \\ S \end{bmatrix}$$

Where, F, a matrix of rates of infection and V, a matrix of rates of transmission, are defined by,

$$F = \begin{bmatrix} 0 & \beta & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, V = \begin{bmatrix} \mu + \alpha & 0 & 0 \\ -\alpha & \gamma + \mu & 0 \\ 0 & 0 & \mu + \beta i \end{bmatrix}$$

The Jacobian matrices of F and V at the disease free equilibrium S(0), E(0), I(0) of the system (1) are respectively

$$DF = \begin{bmatrix} A & 0 \\ 0 & 0 \end{bmatrix}, DV = \begin{bmatrix} B & 0 \\ J1 & J2 \end{bmatrix}$$

$$\text{Where, } A = \begin{bmatrix} 0 & \beta \\ 0 & 0 \end{bmatrix}, B = \begin{bmatrix} \mu + \alpha & 0 \\ -\alpha & \gamma + \mu \end{bmatrix} \text{ and } AB^{-1} = \begin{bmatrix} \frac{\alpha\beta}{(\mu+\alpha)(\gamma+\mu)} & \frac{\beta}{(\gamma+\mu)} \\ 0 & 0 \end{bmatrix}$$

Thus the basic reproduction number is the dominant eigen value of $R_0 = \frac{\alpha\beta}{(\mu+\alpha)(\gamma+\mu)}$ (3)

Disease free equilibrium (E_0):

To study the local stability of the disease free equilibrium, we first find the Jacobian matrix. If we define eq (1) as

$$\begin{aligned} f(t) &= \mu - \mu s(t) - \beta i(t)s(t) \\ g(t) &= \beta s(t)i(t) - (\mu + \alpha)e(t) \\ h(t) &= \alpha e(t) - (\gamma + \mu)i(t) \end{aligned} \tag{4}$$

Then the Jacobian matrix is computed as

$$J = \begin{bmatrix} \frac{\partial f}{\partial s} & \frac{\partial f}{\partial e} & \frac{\partial f}{\partial i} \\ \frac{\partial g}{\partial s} & \frac{\partial g}{\partial e} & \frac{\partial g}{\partial i} \\ \frac{\partial h}{\partial s} & \frac{\partial h}{\partial e} & \frac{\partial h}{\partial i} \end{bmatrix}$$

$$J = \begin{bmatrix} -\mu - \beta i & 0 & -\beta s \\ \beta i & -(\alpha + \mu) & \beta s \\ 0 & \alpha & -(\gamma + \mu) \end{bmatrix}$$

International Journal of Innovative Research in Science, Engineering and Technology

(An ISO 3297: 2007 Certified Organization)

Website: www.ijirset.com

Vol. 6, Issue 6, June 2017

From eq (4) the disease free point is $s, e, i = (1,0,0)$. So,

$$J_0 = \begin{bmatrix} -\mu & 0 & -\beta \\ 0 & -(\alpha + \mu) & \beta \\ 0 & \alpha & -(\gamma + \mu) \end{bmatrix}$$

Now, $Det |J_0 - \lambda I| = 0$

$$= \begin{vmatrix} -\mu - \lambda & 0 & -\beta \\ 0 & -(\alpha + \mu) - \lambda & \beta \\ 0 & \alpha & -(\gamma + \mu) - \lambda \end{vmatrix}$$

$$= \lambda^3 + \lambda^2(\alpha + \gamma + 3\mu) + \lambda[\mu(\alpha + \gamma + 2\mu) + (\alpha + \mu)(\gamma + \mu) - (\alpha\beta)] + \mu[(\alpha + \mu)(\gamma + \mu) - (\alpha\beta)] \quad (5)$$

Let us consider $Det |J_0 - \lambda I| = a_0\lambda^3 + a_1\lambda^2 + a_2\lambda^1 + a_3\lambda^0$,

From (5)

$$a_0 = 1$$

$$a_1 = \alpha + \gamma + 3\mu$$

$$a_2 = \mu(\alpha + \gamma + 2\mu) + (\alpha + \mu)(\gamma + \mu) - (\alpha\beta)$$

$$a_3 = \mu[(\alpha + \mu)(\gamma + \mu) - (\alpha\beta)]$$

$$\text{So, } \forall i \in \{0,3\}; a_i > 0 \ \& \ \frac{a_1 a_2 - a_0 a_3}{a_1} > 0$$

Thus all the Eigen values of the characteristic equation have negative real part which means disease free equilibrium [11]. The disease free stable when $R_0 < 1$ otherwise unstable.

Endemic equilibrium (E^*):

From (3 & 4) the endemic point is

$$s^*, e^*, i^* = \left[\frac{1}{R_0}, \frac{\mu(R_0 - 1)}{R_0(\mu + \alpha)}, \frac{\mu(R_0 - 1)}{\beta} \right]$$

$$J^* = \begin{bmatrix} -\mu R_0 & 0 & -\frac{\beta}{R_0} \\ \mu(R_0 - 1) & -(\alpha + \mu) & \frac{\beta}{R_0} \\ 0 & \alpha & -(\gamma + \mu) \end{bmatrix}$$

Now $Det |J^* - \lambda I| = 0$

International Journal of Innovative Research in Science, Engineering and Technology

(An ISO 3297: 2007 Certified Organization)

Website: www.ijirset.com

Vol. 6, Issue 6, June 2017

$$\begin{aligned}
 &= \begin{vmatrix} -\mu R_0 - \lambda & 0 & -\frac{\beta}{R_0} \\ \mu(R_0 - 1) & -(\alpha + \mu) - \lambda & \frac{\beta}{R_0} \\ 0 & \alpha & -(\gamma + \mu) - \lambda \end{vmatrix} \\
 &= \lambda^3 + \lambda^2[\mu(R_0 + 2) + (\alpha + \gamma)] + \lambda\left[\mu R_0(2\mu + \alpha + \gamma) + (\alpha + \mu)(\gamma + \mu) - \frac{\alpha\beta}{R_0}\right] + \\
 &+ \left[(\mu R_0)(\alpha + \mu)(\gamma + \mu) - \frac{\alpha\beta}{R_0}\mu\right] \quad (6)
 \end{aligned}$$

Let us consider $Det |J^* - \lambda I| = b_0\lambda^3 + b_1\lambda^2 + b_2\lambda^1 + b_3\lambda^0$,
From (6)

$$\begin{aligned}
 b_0 &= 1 \\
 b_1 &= \mu(R_0 + 2) + (\alpha + \gamma) \\
 b_2 &= \mu R_0(2\mu + \alpha + \gamma) + (\alpha + \mu)(\gamma + \mu) - \frac{\alpha\beta}{R_0} \\
 b_3 &= (\mu R_0)(\alpha + \mu)(\gamma + \mu) - \frac{\alpha\beta}{R_0}\mu \\
 \text{So, } \forall i \in \{0,3\}; b_i &> 0 \quad \& \quad \frac{b_1 b_2 - b_0 b_3}{b_1} > 0
 \end{aligned}$$

Thus all the Eigen values of the characteristic equation have negative real part which means a stable equilibrium [11].
And by Routh-Hurwitz criteria the endemic steady state is stable when $R_0 > 1$ otherwise unstable.

IV. CONCLUSION

Even if the calculations of R_0 and E_0 differ, they correlate while interpreting them. If $R_0 < 1$ then the disease will eventually die. In addition, if at any time, R_0 gets smaller than 1, the disease eventually disappears from the population, and the disease-free equilibrium E_0 is globally stable in the feasible region. Mathematically,

$$R_0 \leq 1 \Rightarrow \lim_{t \rightarrow \infty} (s(t), e(t), i(t), r(t)) = (1, 0, 0, 0) = E_0$$

The disease spreads if $R_0 > 1$ and a unique endemic equilibrium (E^*) is globally asymptotically stable in the interior of the feasible region and the disease will persist at the endemic equilibrium if it is initially present, i.e. the epidemic builds up [12-14]. Mathematically,

$$R_0 \geq 1 \Rightarrow \lim_{t \rightarrow \infty} (s(t), e(t), i(t), r(t)) = E^*$$

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Vol. 6, Issue 6, June 2017

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